FOCAL DEGENERATION OF THE LUMBAR CORD IN A CASE OF INFANTILE SCURVY

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The number of necropsies on cases of infantile scurvy has not been large. It is a disorder for which we possess a most efficient cure, so that the mortality is low, and the deaths are generally due to some intercurrent disease. It is remarkable to note in reviewing the necropsy protocols, how intensely the interest has been centered on the osseous system. We find the pathologist devoting page after page to the minutiae of the changes in bones, and in most instances either failing to mention the other organs, or passing them by with scant notice. As far as I am aware, there has been no microscopic examination of the nervous system in a case of infantile scurvy. A study of this kind would seem to be indicated in view of the similarity in many of the clinical symptoms between this disease and beriberi, which is classed by some among the diseases of the nervous system. Furthermore, nervous manifestations have been noted in the course of infantile scurvy, such as tachycardia, changes in the knee reflexes, alterations in the optic disks,[1] etc., so that it seemed well worth while to investigate whether the nervous system was involved in this disorder.

Bertha H., aged 18 months, was born and brought up in the Misericordia Hospital of this city. She had never been nursed, but was fed on pasteurized milk, and in addition received cereal gruels. Jan. 3, 1917, she was transferred to the Willard Parker Hospital for infectious diseases, on account of a nasal culture showing the diphtheria bacillus. The throat culture was negative, and there were no clinical signs of diphtheria. She also had marked rickets and scurvy. The latter disease was evident from the subperiosteal swelling of the left humerus, accompanied by a separation of the epiphysis, and swelling of the forearm, from the swelling and tenderness of the right thigh, the edema of both legs and feet, and from a subcutaneous hemorrhage of the eyelid.

She had received orange juice and beef juice for some days, and this treatment was continued during the stay at the hospital. However, the temperature was 103-104 F., and soon definite signs of pneumonia developed, of which disease she died Jan. 14. The child was poorly developed and had a marked

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[1] Hess, A. F.: Jour. Am. Med. Assn., 1917, 68, p. 235.

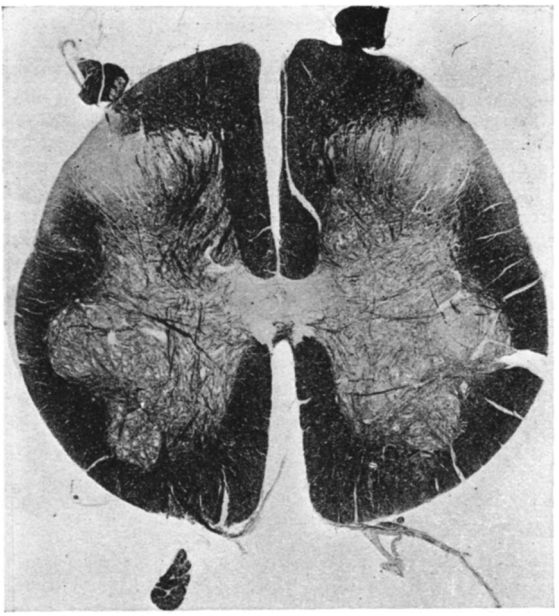

Fig. 1.—Low power. Showing pale area and paucity of cells and fibers in lateral group of left anterior horn. (Frozen section, Spielmeyer's iron alum hematoxylin method.)

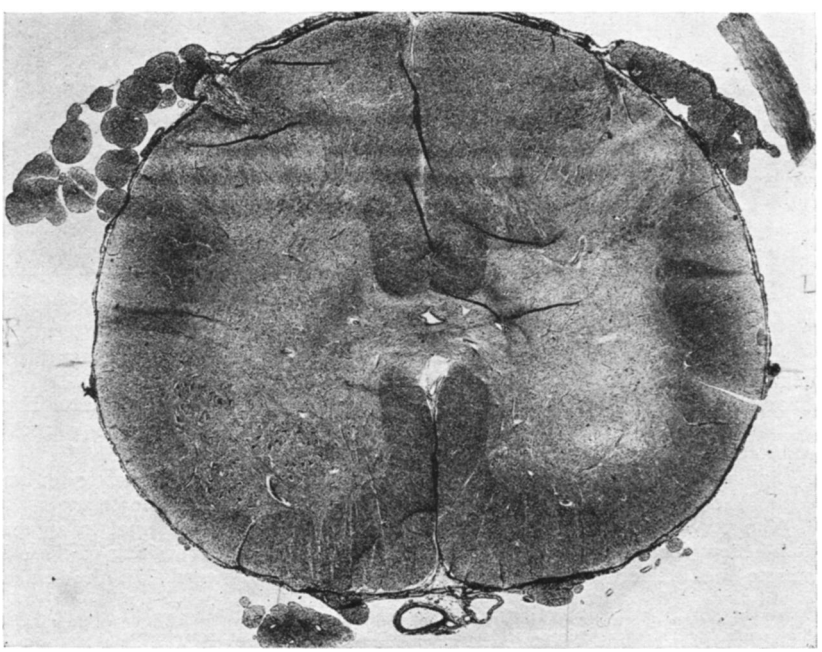

Fig. 2.—Lower power. Showing same as previous figure. (Marchi stain, vital scharlach counter stain.)

rachitic rosary. The plasma coagulation time was normal (6 minutes), bleeding time normal, "capillary resistance test" negative, puncture test positive.

At necropsy, separation of the epiphysis of the left humerus was found. There had evidently also been an infraction of the head of the right femur, so that it was depressed, and assumed a position almost at right angles with the shaft of the bone—a condition closely resembling that of coxa vara.* There were petechial hemorrhages in the pericardium with some slight increase of its fluid contents. The heart was slightly enlarged, the lungs showed a considerable pneumonic involvement of the right upper and left lower lobes; the abdominal viscera showed no changes excepting a few petechiae in both kidneys.

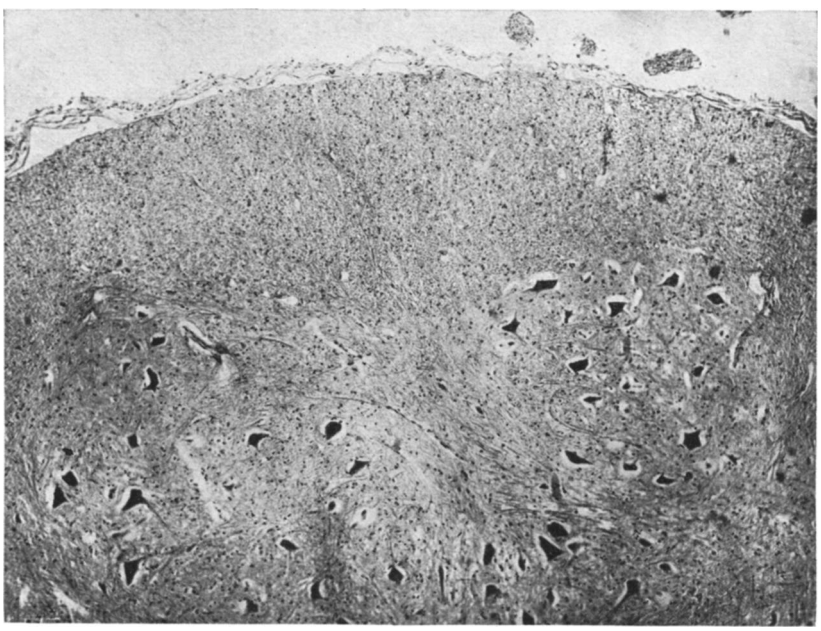

Fig. 3.—High power. Showing normal cells in lateral group of right anterior horn.

As mentioned, special attention was given to the spinal cord and peripheral nerves. The tissues were preserved in formalin, and were prepared by Dr. Charles B. Dunlap of the Psychiatric Institute of the New York State Hospital, whom I take this opportunity to thank for his painstaking work in this connection. The following is a summary of his report:

Spinal Cord: Sections were stained with toluidin blue, hematoxylin and eosin, Weigert's neuroglia, Marchi, scharlach R. and myelin sheath stains.

The nerve cells and neuroglia in the cervical part were normal in appearance except for a slight excess of neuroglia cells, and some bunching of these in minute foci. There were no changes in the middorsal cord. In the lumbar

* This lesion of coxa vara in association with infantile scurvy is of interest in view of the possibility that it may, in some instances, be the cause of this malformation.

cord there was a striking local loss of nerve cells in the lateral groups of the left anterior horn. A pale area was seen at this site containing numerous irregular nuclei. The preserved nerve cells were in fair condition. The pia contained more cells than usual, but not a pronounced exudate. The neuroglia was about equal on both sides. The Marchi stain was negative, but scharlach R. showed abundant fat droplets in the pale area and along the course of the anterior root fibers. There were fewer nerve fibers in the pale area than on the right, but less difference than one would expect. At slightly different

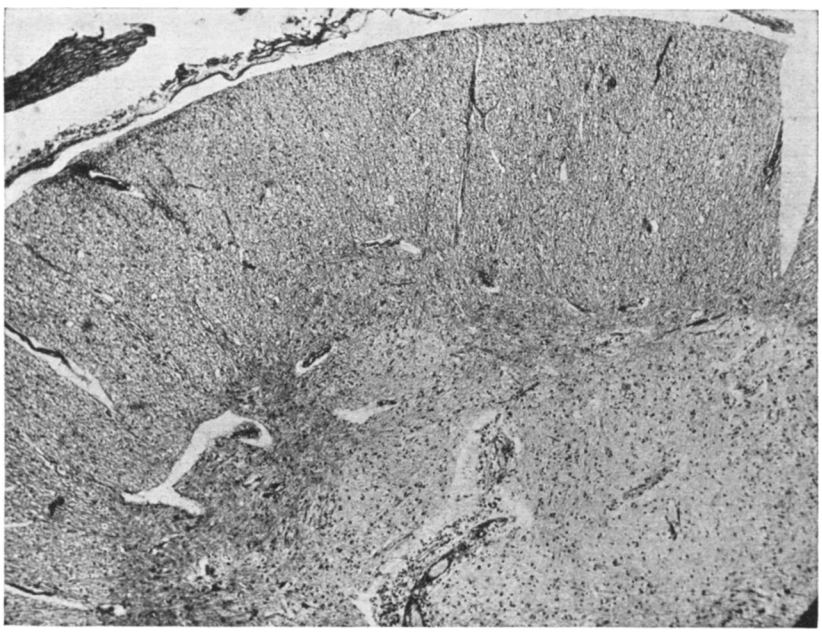

Fig. 4.—High power. Showing focal degeneration and absence of cells in lateral group of left anterior horn.

levels on the left, the nerve cells increased, indicating the local nature of the lesion. The cell counts on the two sides, as observed with various stains, were as follows:

	General Averages	
	Lesion Side	Sound Side
Alcohol	11	50
Neuroglia	11	34
Weigert sheath	26	36
Marchi	11	43

Brachial plexus was normal except for pale staining of the myelin sheaths.

Transverse and longitudinal sections of the sciatic nerve failed to show any changes except pallor. There were numerous bundles—more than 50.

The pneumogastric nerve, upper portion, in transverse and longitudinal sections appeared normal except for pallor of the nerve sheaths, and many small rings in which no axis cylinders were seen. In the lower portion was possibly a slight exudate in the epineural sheath and connective tissue.

The examination may be summarized by the statement that the only definite change found was a focal degeneration of the lumbar cord, which extended for a distance of perhaps a quarter inch. We have no means of definitely interpreting these changes. That they were not the result of poliomyelitis would seem evident, not only on account of the noninflammatory nature of the lesion with its absence of round cell infiltration, and lack of characteristic changes in the remaining anterior horn cells, but also in view of the fact that the child had never shown any symptoms of this disease, and had been brought up in an institution where no instance of infantile paralysis had developed. It would seem to belong rather to the degenerative type of lesion. Whether it is truly scorbutic in nature or merely happened to be associated with this disease, only an examination of other cases can decide. In some respects it resembles the microscopic lesions of lead poisoning, described by Oppenheim and Monakow, and similar lesions reported in experimental lead poisoning by Stieglitz.[1] In this connection it is of interest to remember that Eijkmann,[2] in his classic article first describing the polyneuritis of fowl, mentions changes in the cord, "especially degenerative and atrophic changes in the ganglion cells of the anterior horns," and that Shiga and Kusama[3] substantiated this observation by finding a similar atrophy of the ganglion cells of the anterior horns. My case is reported rather to stimulate further investigation than to pronounce definite judgment on the proper interpretation of the cord lesion.

[1] Lewandowsky: Handb. d. Neurologie, 1911, 2, p. 55.
[2] Virchow's Arch., 1897, 148, p. 523.
[3] Beihefte z. Arch. f. Schiffs- u. Tropen-Hyg., 1911, 15, Part 3.